# ROLE OF ENDOSCOPY IN FOLLOW UP OF PATIENTS UNDERWENT SURGERY FOR DUODENAL ULCER PERFORATION

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## BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"ROLE OF ENDOSCOPY IN FOLLOW UP OF PATIENTS UNDERWENT SURGERY FOR DUODENAL ULCER PERFORATION"** is bonafide record work done by **Dr. K. SIVAKUMAR** under my direct supervision and guidance, submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment of University regulation for M.S. General Surgery, Branch I.


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## DECLARATION

I **Dr. K. SIVAKUMAR** solemnly declare that the dissertation titled **"ROLE OF ENDOSCOPY IN FOLLOW UP OF PATIENTS UNDERWENT SURGERY FOR DUODENAL ULCER PERFORATION"** has been prepared by me. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other University board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of M.S.(General Surgery) Branch – I to be held in March 2008.

**Place :** Madurai **Dr.K. SIVAKUMAR**

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# CONTENTS

# INTRODUCTION

Elective operations for peptic ulcer were among commonest procedures by general surgeons in the middle decades of the last century. The operations were designated to reduce acid secretions of the stomach and their role become increasingly unimportant as more potent antisecretory drugs like H2 receptors antagonists and proton pump inhibitors become available.

Initially there was concern regarding any long term side effects of the new drugs and for some years surgery was still advised for younger, fitter patients to avoid a lifetime medication.

The identification of Helicobator pylori as a causative agent combined with effective regimens for its eradication further changed surgical practice. The majority of the patient could be cured, did not require long term acid suppression.

Surgery is mainly confined to the management of complications of peptic ulcer disease including perforation, haemorrhage and gastric outlet obstruction. Surgery is also occasionally indicated for ulceration refractory to medical treatment and for complications of previous peptic ulcer surgery.

## AIM OF THE STUDY

Endoscopic evaluation in reviewing patients who had undergone duodenal ulcer perforation closure with omental patch and to deside need for definitive surgery.

# REVIEW OF LITERATURE

## ANATOMY:

The duodenum is closely associated with head of pancreas and is divided in to four parts. The first part extend from pyloric sphincter is about 5 cm long. It passes backward and to the right and its final portion is fixed to the posterior abdominal wall. The Hepatic artery passes along its upper border and the gastro duodenal artery, portal vein and common bile duct pass behind it.

The second part is 10 cm long and descends vertically on the right side of vertebral column to the level of L3 or L4. It is crossed by the root of the transverse mesocolon. Its medial wall receives common bile duct and main pancreatic duct. Posteriorly it is related to the right kidney and inferior vena cava.

The third part of the duodenum is 7.5 cm long, extend horizontally from right to left side of L3. Anteriorly it is crossed by superior mesenteric vessels and root of mesentery and is posteriorly related to interior vena cava and aorta.

The fourth part of duodenum is 2.5 to 5 cm long and ascends on the left side of aorta to the level of L2 where it becomes duodeno

jejunal flexure. Several recesses are found around this flexure and on rare occasions they are the sites of internal herniation.

**BLOOD SUPPLY:**

The arteries are derived from the coeliac and superior mesenteric vessel.

The first part receives branches from the right gastric, gastro epiploic arteries. The superior pancreatico duodenal artery, a branch of gastro duodenal artery runs downward in the concavity of the duodenum to anastomose with inferior pancreaticoduodenal branch of the superior mesenteric artery. The veins accompany the arteries and drain into the portal system via the splenic or superior mesenteric veins.

**LYMPHATIC DRAINAGE :**

The lymphatics of the duodenum anastomose freely within the submucosa. Those of the anterior collecting vessel pass to nodes along the anterior branch of superior pancreaticoduodenal artery and drain upward to the subpyloric and celiac nodes. The posterior collecting vessels pass to nodes behind the head of pancreas and

drain the pancreatico duodenal nodes associated with the superior mesenteric vessels.

**PEPTIC ULCER:**

Peptic Ulcer refers to a group of ulcerative disorders of the upper gastrointestinal tract involving principally the most proximal portion of duodenum and stomach which has in common the participation of acid-pepsin in their pathogenesis. This includes duodenal and gastric ulcer and also ulcers associated with Zollinger – Ellison syndrome caused by gastrinoma and also ulcers associated with stress or drug ingestion. Duodenal ulcer is characteristically a chronic and recurrent disease.

Ulcer development or resistance to ulceration depends on balance between aggressive factors and mucosal factors. Peptic ulcer results when aggressive effects of acid – pepsin over weight the protective effect of gastric or duodenal mucosal defence. Proteolytic effects of pepsin in concert with the corrosive properties of secreted gastric acid are the integral component which amount for tissue injury that produces peptic ulcer. Activity of pepsin is maximum at a pH of 2.0 (approx.) and reduced substantially above pH 4.

Mucosal injury from asprin, NSAID and bile acid is augmented in presence of acid. On the other hand ethanol causes mucosal injury with or without acid. Corticosteroids, smoking, psychological and physiological stress appears to predispose to mucosal injury by some mechanism incompletely understood.

## PHYSIOLOGY RELATED TO PEPTIC ULCER

1. Aggressive factors: Acid (Hcl) secretion by parietal (oxyntic) cells involves a process of oxidative phosphorylation. Hydrogen ion secreted accompanied by chloride-ion and with increase in Hydrogen ion there is reciprocal decrease in Sodium ion secretion. For each Hydrogen ion secreted into the gastric lumen one bicarbonate ($HCO3^-$) is released into gastric venous circulation accounting for the ‘alkaline tide’ a direct reflection of magnitude of gastric Hydrogen ion secretion. Bicarbonate is released from the carbonic acid generated from carbon dioxide by parietal cell carbonic anhydrase. The final step in hydrogen ion secretion is accomplished by proton pump mechanism involving H+ K+ ATPase located in apical microvillus membrane and tubovesiculur apparatus of the parietal cell.

Regulation of gastric acid secretion involves chemical, neural or hormonal factors. Gastric acid secretion is stimulated by gastrin, post ganglionic vagal fibers via muscarinic-cholinergic receptors of parietal cells.

**GASTRIN:**

Gastrin the most potent stimulant of gastric acid secretion is contained and released from Gastrin cells (G-cells scattered singly or in small clusters among the epithelial lining cells in middle and deeper portion of antral pyloric glands.)

Gastrin release is stimulated by a neuropeptide–gastrin releasing pepitide and the principal form in gastric antral mucosa and gastrin is heptadecapeptide G17 (17 Aminoacid residue with active site at carboxyterminal tetrapeptide amide). Gastrin 17 has shorter half –life than G34, is as potent as G34 in stimulation of acid secretion. Most proximal duodenal mucosa has 10% of antral concentration. Gastrin stimulates gastric acid secretion by direct stimulation of parietal cells and by stimulating histamine release by ‘E.C.L’ cells (Entero – Chromaffin like cells).

**VAGAL STIMULATION:**

Increases the gastric acid secretion by cholinergic stimulation of parietal cell and causes release of gastrin into circulation. It lowers parietal cell threshold in response to circulating gastrin concentration. Certain vagal branches or fibres also inhibit gastrin release.

HISTAMINE:

Gastric mucosa contains large amount of histamine. Histamine is contained in cytoplasmic granules of mast cells, which are interstitial in location and Entero chromffin like (E.C.L.) cells distributed singly in oxyntic glands often in direct contact with parietal cell.

Histamine plays an important role in stimulating gastric acid secretion. It acts in concert with cholinergic activity on parietal cells. There exists uncertainly whether histamine is final common effector molecule in stimulation of parietal cell secretion.

Histamine stimulates gastric acid secretion by increasing parietal cell cyclic AMP thereby activating cyclic AMP dependent protein kinases.

Gastrin & Acetylcholine do not stimulate cAMP production, stimulate acid secretion by increasing parietal cells cytosolic calcium.

## REGULATION OF GASTRIC ACID SECRETION

This includes:

i. Cephalic phase : Response to sight, smell, taste and anticipation of food.

ii. Gastric phase : Induced by food in stomach &

iii. Intestinal phase : Due to entry or presence of food within the lumen of small intestine.

Basal or inter digestic gastric acid secretion is considered to be a fourth phase of acid secretion.

Cephalic phase include cortical and hypothalamic components and mediated primarily by vagal stimulation.

Gastric phase is effected by food principally protein and products of protein digestion by increasing gastrin release. Oral glucose or fat causes slight increase in serum gastrin but not gastric acid secretion.

INHIBITION OF GASTRIC ACID SECRETION:

Acid in stomach or duodenum, hyperglycemia, hypertonic fluids or fat in duodenum inhibit gastric secretion. Reduction of intragastric pH to 3.0 produces inhibition of gastrin release and pH of 1.5 blocks completely.

Somatostatin plays an important role in acid induced feed back control inhibition of gastrin release (paracrine effect on gastrin cells).

Secretin, linear polypeptide (27 AA ) is capable of inhibiting gastric acid secretion.

**CONCEPTS OF PROTECTION:**

**1. Gastric mucosal barrier concept : (Horace Davenport)**

This describes the ability of gastric mucosa to resist back diffusion of Hydrogen (H+) ion and thus to contain a high concentration of Hydrochloric acid within the gastric lumen. When this barrier is broken by an injurious agent H+ defuses rapidly back into the mucosa, which results in mucosal injury.

## 2. Concept of cytoprotection:

The term cytoprotection is a misnomer in that it does not refer to protection of individual cells but rather to protection of deeper layers of mucosa against injury.

**MEDIATOR OF MUCOSAL PROTECTION:**

**a. Mucus:** Secreted by surface epithelial cells physically protects the mucosa from abrasions. It also resists the passage of large molecules such as pepsin, but H+ and other small particles like ethanol can easily penetrate it.

**b. Bicarbonate:** Produced in small amount by surface epithelial cells and also diffuses from mucosa creating thin layer of alkalinity between mucosa and epithelial cells.

**c. Hydrophobic layer of phospholipid** coats the luminal membrane of surface epithelial cells.

**d. Mucosal blood flow:** Mucosal blood flow maintains oxygenation and supply of nutrients and dispose absorbed acid and noxious agents.

**e. Alkaline tide:** This refers to mild alkalination of blood and mucosa that results from the secretion of a molecule of $HCO3^-$ by

parietal cells into the adjacent mucosa for every H+ that is secreted into the lumen.

**f. Epithelial renewal:** Proliferation of new cells with differentiation and migration to replace old cells.

**g. Restitution** refers to phenomina of rapid migration of cells within the mucosa to cover denuded surface epithelium (rapid healing of small areas of superficial injury).

**h. Prostoglandins** particularly E &I stimulate mucus and bicarbonate secretion, maintain mucosal blood flow and have protective effect on epithelial cells.

**i. Epithelial growth factor (EGF)** secreted by saliva & duodenal mucosa has a protective effect on gastroduodenal mucosa.

## ASSOCIATION OF H PYLORI AND PEPTIC ULCER

It is found that H pylori is present in 90% patients with duodenal ulcer and 75% patients with gastric ulcer infection appears to be acquired in childhood and is inversely associated with socio economic status.

H pyloric is microaerophilic spiral or helical gram negative rod with 4-6 flagella. It resides in gastric type epithelium with in or beneath the mucus layer. It is one of the most potent producers of crease enzyme that is capable of splitting area into ammonia and bicarbonate.

H. pylori induced gastro intestinal injury remain to be fully elucidated.

Three potential mechanisms have been proposed.

i. Production of toxic products to cause local tissue injury.
ii. Induction of a local mucosal immune response.
iii. Increased gastrin level with a resultant increase in acid secretion.

The mucosal barrier is disturbed by the local immune response and the generation of large amount leads to alteration in pH, mucosal charge gradient, cellular permeability, and epithelial sodium potassium ATPase activity leading to increased hydrogen ions.

**Diagnostic tests for H.pylori**

- Non-invasive tests include serology and carbon labeled urea breath test.
- Invasive tests include rapid urease test, histology and culture but require the employment of endoscopy.
- Special stains such as Giemsa and Warthin starry silver stain are used for improved visibility than routine eosin and hematoxylin.

H.pylori infections show a strong association with peptic ulcer perforation. Eradication therapy directed against H.pylori promotes ulcer healing and prevents recurrence.

The original triple anti H.pylori therapy used Bismuth Salt (Bismuth subsalicylate 5 –tablets daily, along with Tetracycline or Ampicillin 500 mg bid and metronidazole 250 mg bid for two weeks and Ranitidin 300 mg until healing.

The Newer regimen using a proton pump inhibitor (eg. Omeprazole) with two antibiotics twice daily have, an eradication of 90% that can be achieved with 1 week treatment.

Only patients who fail to achieve eradication after multiple attempts and those who are the H.pylori negative should be considered for surgery. Recurrent ulcer and bleeding can be prevented by H pylori eradication. Eradication therapy has also been shown to be superior to long term maintenance with H2 receptor antagonist in the long term management of patients with bleeding ulcers.

## DUODENAL ULCER & PERFORATION

Duodenal ulcer more frequently involves middle age group and highest incidence of perforation is between 45-55 yrs of age. But duodenal ulcer disease is reported in children also, with positive family history of peptic ulcer, blood group ‘O’ and regional diet were considered predisposing factors 1 and duodenal ulcer perforation was reported in 9 yrs old child which is not usually considered in differential diagnosis of acute abdomen in the children. Male: Female ratio varies between 2:1 to 4:1. Perforation is the commonest complication of duodenal ulcer. The incidence is 7 to 10 case per 100 000 people / year.

Patients with perforation of acute ulcers (20%) usually gives short lived attacks of dyspepsia or may be ‘silent’. Chronic duodenal ulcer patients (80%) may have genetic back ground (MEN syndrome) and blood group ‘O’ are three times likely to develop it.

A study of rhythmic patterns in incidence of peptic ulcer perforation showed a circadian rhythm (daily) was found in younger patients, males and duodenal perforations while 12 hr (circasemedian) rhythm characterized ulcer perforation for women and gastric ulcers. Duodenal perforation showed highest incidence in the afternoon, while gastric perforation showed a major peak around noon and a secondary peak near midnight.

- Now increasing use of NSAIDs have resulted in a shift in the incidence of perforation in the $6^{th}$ and $7^{th}$ decade of life.
- Peptic ulcer perforation are more common in patients of low socio economic status.

RISK FACTORS

1. Use of NSAID
2. Smoking
3. Increasing patients age

4. Patients on immuno suppressive therapy
5. Chronic obstructive pulmonary disease
6. Major burns
7. Multi organ system failure.

## PATHOPHYSIOLOGY

A peptic ulcer is said to have perforated when it extends through the muscle wall and serosa of the gastro intestinal tract thereby establishing communication between the lumen and adjacent space or structure. The perforation occurs as a result of sudden sloughing of the base of the ulcer due to impaired blood supply.

The site of pyloroduodenal perforations is usually the anterior wall and majority of the perforated gastric ulcers are located on the lesser curvature. Posterior perforation of a gastric ulcer may occur into the lesser sac.

Perforation leads to leakage of gastric or duodenal contents into the peritoneal cavity initiating an acute peritonitis. Although it is an initial chemical peritonitis, bacterial peritonitis supervenes over the next few hours.

The presence of bacteria in the peritoneal cavity stimulates an inflow of acute inflammatory cells. The omentum and the viscera tend to localize the site of inflammation. This results in an area of localized hypoxia, which in turn facilitates growth of anaeraobes and produce impairment of bactericidal activity of granulocytes. This leads to increased phagocytic activity of granulocyte, degradation of cells, hypersecretion of fluid forming the abscess, osmotic effects shift of more fluids into the abscess area and enlargement of the peritoneal exudates causing paralytic ileus.

Absorption of bacterial endotoxins through the inflamed peritoneal surface causes endotoxemia. The combination of fluid and electrolyte imbalance and septicemia results on shock and multi organ failure, which is the cause of, increased mortality in untreated patients of perforative peritonitis.

**MICROBIOLOGY**

The microbiology of the gastro intestinal tract changes from its proximal to distal part. Few bacteria populate the proximal part of the bowel, where as the distal bowel contains aerobic organisms and

higher percentage of anaerobic organisms. The common organisms are Escherichia coli and Bacteroides fragilis.

CLINICAL FEATURES

Peptic ulcer perforation are classified into

- Acute free perforation
- Confined perforation / chronic penetration.

ACUTE FREE PERFORATION

The clinical course is classically divided into 3 stages.

- Primary stage – Stage of peritoneal irritation.
- Secondary stage – Stage of peritoneal reaction
- Tertiary stage – Stage of bacterial peritonitis.

STAGE OF PERITONEAL IRRITATION

Also known as peritonism. This stage lasts for the first 2 to 3 hours following perforation. The sudden outpouring of caustic gastric juice into the peritoneal cavity producing chemical peritonitis causes the initial symptoms. The patient can recall the exact time of perforation by the abrupt on set of intense abdominal pain. The patient may or may not vomit. Referred pain is felt over the tip of left shoulder in 1/3 to 1/2 of the patients due to irritation under the

dome of diaphragm. Initially the patient may be shocked with a tachycardia but there is the little change in the temperature. Respiration is shallow and the abdomen does not move with respiration. Tenderness and muscle guarding are constantly present over the right side of the abdomen.

## STAGE OF PERITONEAL REACTION

During the secondary stage, the gastric juice is diluted by the peritoneal exudates. The patient feels comfortable due to the buffering action of the fluid secreted. Symptoms are reduced but signs are still present. Muscular rigidity continues to be present. This stage is marked by two other features; obliteration of liver dullness and presence of shifting dullness. Evidence of free air within the abdominal cavity may be seen on  a plain upright radiograph of the abdomen and chest in nearly 70% of the cases.

## STAGE OF DIFFUSE PERITONITIS

In the tertiary stage, with the establishment of bacterial peritonitis, the patient has gone a step further towards the grave. The

pinched and anxious face, sunken eyes and hollow cheeks – so called facies hippocratica, with rising pulse rate which is low in volume and tension, persistent vomiting, board like rigidity of the abdomen, increasing the distension of the abdomen all are evident in the terminal stage.

At times the spillage of the luminal contents is more of seepage and if seepage becomes contained in a smaller area, the pain though intensive, is located near the site of perforation and muscular rigidity is limited in extent. In posterior perforation the inflammatory reaction is contained in the lesser sac and symptoms may be obscure.

**FORME FRUSTE**

When the breach in the wall of the stomach or duodenum is small and spontaneously sealed off rapidly before contamination of the general peritoneal cavity pain is less intense. Guarding and rigidity is present only in the epigastrium and right hypochondrium. Other areas of the abdomen are soft; this condition is known as forme fruste of acute free perforation.

## CONFINED PERFORATION / GRADUAL PENETRATION

Penetration occurs when a peptic ulcer burrows through the wall of the stomach or duodenum but, instead of perforating freely into the peritoneal cavity, the crater bores into an adjacent organ. Duodenal ulcers that involve the posterior wall of the bulb can penetrate into the pancreas. Rarely, penetrating peptic ulcers can result in the development of fistula between the duodenum and the common bile duct (choledochoduodenal fistula).

Penetration can be associated with a change in the typical pattern of ulcer symptoms, patient may complain of an increasing intensity or longer duration of pain, or they may notice that the pain radiates into the back or that eating no longer relieves the discomfort.

## PERFORATION AND HAEMORRHAGE

The combination of perforation and hemorrhage occurs in either way.

1. Perforation occurring in the course of medical management of hemorrhage.
2. Onset of hemorrhage after a recent perforation.

## INVESTIGATIONS

1. IMAGINE STUDIES

a) X rays

i. Erect radiographs of the chest and a plain upright radiograph of the abdomen are the most common first line of diagnostic imaging when a perforated peptic ulcer is considered.

As little as 1 ml of free air may be visualized. Free air is present in 6% to 80% of cases. In the upright view, curvilinear lucencies separate the most superior portion of the diaphragm from the liver on the right side and from the stomach and spleen on the left. An air-fluid level in the stomach should not be mistaken for free air. Usually the lateral margin of the air-fluid level can be seen extending to the lateral wall of the stomach, demarcated by serosal fat.

**Causes of pseudopneumo peritoneum in a plain X ray Abdomen are**

- Chilladiti syndrome
- Sub diaphragmatic fat
- Curvilinear pulmonary collapse
- Omental fat

- Subphrenic abscess with gas forming organisms
- Subpulmonary pneumothroax
- Intramural gas in pneumatosis intestinalis

ii) On the lateral decubitus view, the free air is usually best seen adjacent to the lateral margin of the liver, but in some patients the iliac portions of the peritoneum are more superior in location and free gas accumulates preferentially over the upper iliac bone.

iii) The supine view may occasionally be the only view ordered and available, especially if penumoperitoneum is not suspected,. Pneumoperitoneum can be detected in the supine view if free gas surrounds, a gas-filled bowel loop. In this situation, the inner and outer margins of bowel wall are clearly seen (the Rigler sign). Some fat may normally outline the serosal surface of bowel loops, but in the presence of pneumoperitoneum the outer surface of the bowel is sharply marginated and more distinct than fat – outlined bowel. Small amounts of air rise to the most superior portions of the abdomen and may be seen outlining the anterior margin of the

liver, forming an oblique or triangular lucency superimposed over the lower portion of the liver. A linear lucency overlying the medial mid-liver may represent free air in the fissure for the ligamentum teres. If large amounts of free air are present, air may outline the falciform ligament anterior to the liver, producing the “football” sign, a large oval collection of air with a central soft tissue stripe produced by the falciform ligament outlined by surrounding gas. Air under the inferior abdominal wall may outline the umbilical folds the inverted –V sign. The Rigler sign and air collection overlying the liver are the most common signs of free air on a supine abdominal view.

**B) Contract Radiography**

i. Contract radiography using water-soluble diatrizoate meglumine (Gastrograffin ) is useful in doubtful cases. In free perforation there is leakage of contrast into the peritoneal cavity.

ii. Gastrograffin administered contrast is also useful in diagnosis of sealed perforation to plan a conservative management as in the case of forme fruste.

**C) Ultrasonograms of the Abdomen**

Localized gas collection related to bowel perforation may be detectable, particularly if it is associated with other sonographic abnormalities (e.g. thickened bowel loop).

The site of bowel perforation can be detected by sonography (e.g. gastric vs. duodenal perforation)

Ultrasonograms of the abdomen can also provide rapid evaluation of the liver, spleen, pancreas, kidneys, ovaries, adrenals and uterus, to rule out associated pathology.

D) CT Scans of the Abdomen

This modality can be a valuable investigative tool, providing differential morphologic information not obtainable with plain radiography or ultrasonography.

CT Scans may provide evidence of localized perforation(e.g., perforated duodenal ulcer ) with leakage in the area of the gallbladder and right flank with or without free air being apparent.

**e) Radionuclide scan**

Detection of perforation using 99 m Tc labeled sulfur with water & 99mTc DISIDA were reported.

**2. LAB STUDIES**

**Complete Hemogram:**

Parameters suggestive of infection (e.g.,leukocytosis); Leukocytosis may be absent in elderly patients.

- Elevated packed blood cell volume suggests a shift of intravascular fluid.
- Blood culture for aerobic and anaerobic organisms.
- Liver function and renal function: Findings may be within reference ranges, when no preexisting disorder is present.

**3. Other Tests :**

Laparoscopy improves surgical decision making in patients with acute abdominal pain, particularly when the need for operation is uncertain.

**DIFFERENTIAL DIAGNOSIS**

- Acute appendicitis
- Cholecystitits, biliary colic

- Acute pancreatitits
- Typhoid fever
- Meckel’s diverticulum
- Diverticular disease
- Ischemic colitis
- Inflammatory bowel disease
- Colitis
- Acute salphingitis
- Endometriosis
- Pelvic inflammatory disease
- Ovarian torsion
- Constipation

**The non – abdominal conditions resembling perforation are**

- Myocardial infarction
- Pleurisy
- Spontaneous pneumothroax
- Diabetes mellitus
- Acute porphyria

MANAGEMENT

Divided into conservative and operative management.

**CONSERVATIVE MANAGEMENT**

There are several studies advocating non-operative management in selected patients with a successful outcome. The candidates who are tolerating the insult well and in whom perforation seems to have sealed, can be managed conservatively. Resuscitation with intravenous fluids, nasogastric suction and intravenous antibiotics and H2 blockers resulted in mortality and mobility similar to those of operative management, but hospitalization is prolonged and incidence of subphrenic abscess is high. If non operative treatment is chosen then the patient will require frequent clinical evaluation, so that operative therapy can be initiated at the first sign of clinical deterioration.

**PRE-OPERATIVE MANAGEMENT**

The initial priorities are resuscitation and analgesia.

- Correction of fluid and electrolyte imbalance: Extracellular fluid losses are replaced by colloids or crystalloids that have an electrolyte composition similar to plasma.

- Monitoring of Central venous pressure (CVP) in critically ill and / or elderly patients, in whom cardiac impairment may be exacerbated by large fluid loss.
- Administration of systemic antibiotics and establishing the likely organisms.
- Nasogastric suction to empty the stomach and reduce the risk of further vomiting.
- Urinary catheterization to assess urinary flow and adequacy of fluid replacement.
- Analgesics, such as morphine, in small intravenous doses, preferably as a continuous infusion.

OPERATIVE MANAGEMENT

1. Simple closure with dead omental patch.
2. Simple closure with live omental patch.
3. Simple closure with definitive procedure for ulcer.
4. Endoscopic closure of perforated ulcer.
5. Laparoscopic closure of perforated ulcer.
6. Closure with serosal patch

These are the various target oriented operative techniques. All these techniques should be supplemented with thorough peritoneal lavage. Laparoscopic approach holds good in peritoneal lavage permitting irrigation of all corners of the peritoneal cavity.

7. Flank drain and conservative management is a non target oriented technique in patients of poor general conditions.

**DEFINITIVE PROCEDURES FOR DUODENAL ULCER PERFORATION**

1) Truncal vagotomy with suitable drainage procedure
2) Highly selective vagotomy
3) Taylor's procedure (anterior seromyotomy with posterior truncal vagotomy)

**Investigations for follow up :**

**1. Contrast Radiography :**

For decades the mainstay of gastro intestinal diagnosis was contrast radiography. The routine examination of esophagus, stomach and duodenum has largely been displaced by flexible endoscopy. Radiologic examination offers unique advantage with ability to identify diverticula, fistula and anastomatic leak more rapid with contrast radiography than endoscopy. In addition contrast radiography can be complementary to endoscopy in the assessment of complicated luminal strictures in esophagus, gastro oesophageal junction and duodenum.

When there is concern of barium soilage due to leak, water soluble contrast medium should be used.

Contrast radiography of upper gastrointestinal tract has been displaced by endoscopy in the evaluation of the bleeding.

Angiography may be useful in case occult bleeding in post operative haemorrhage and in torrential upper gastro intestinal bleeding when endoscopy is not possible.

## 2. Gastrointestinal Endoscopy :

Since 1960, instrumentation and technique of flexible endoscopic examination of gastro intestinal tract have established was dominant modality for the diagnosis of gastrointestinal disease. The therapeutic potential of flexible endoscopy is rapidly expanding. Duodenal ulcer is rarely associated with malignancy so biopsies are not routinely required. Bleeding duodenal ulcer may demonstrate 'visible vessel' which portends high likelihood of recurrent bleeding. Endoscopic therapy with infection of epinephrine or the use of thermal probes or lasers have been used in control of haemorrhage from these ulcers.

Duodenitis may be mild or severe and commonly involved diffuse mucosal erythema and friability.

Gaint duodenal ulcer, defined as ulcer more than 2 cm in diameter is usually found in posterior aspect of duodenal bulb penetrating into the pancreas where it is associated with a significant risk of bleeding from underlying, gastro duodenal artery. In addition to providing diagnosis, endoscopy provides ability to sample tissue for H.pylori testing.

Gastric emptying scans (GESs) useful in evaluating patient with suspected gastric dysmotility

**Gastroduodenal monometry :**

Intraluminal gastro jejunal pressure recording or GDM is useful in patient having nausea, vomiting and duodenal pain unexplained by GES and structural testing to determine whether small bowel stasis is neuropathic or myopathic process and for patients with suspected chronic intestinal pseudoobstruction.

**Gastric Analysis :**

Gastric secretion analysis and the direct measurement of gastric acids are limited to the evaluation of patients with elevated serum gastrin and suspected hypersecretory syndromes. Under basal conditions normal patients will produce upto 2 mEq acid/hr where as the average secretion in patients with duodenal ulcer is approximately 4 mEq / hr.

If Basal acid output(BAO) is more than 10 mEq / hr Zolinger – Ellison syndrome (gastrinoma) should be considered.

The diagnosis is more likely if BAO approaches the Maximal acid output (ie. Ratio of BAO to MO is 0.6 or greater)

S. gastrin : Serum gastrin determination is indicated for patients who fails to respond to H.Pylori eradiction. Fasting S. Gastrin more than 1000pg/ml is highly suggestive of ZES.

# LAPAROSCOPIC MANAGEMENT OF PERFORATED DUODENAL ULCER

For patients with perforated ulcer the conventional treatment is laparotomy and an omental patch repair. Simple closure with or without the $H_2$ receptor antagonist is the most popular method of management of perforated acute duodenal ulcer.

With laparascopic revolution, laparascopic omental patch repair has become relatively easy and should be within the compliance of general surgeons.

It is ideal to select the stable patient and peritonitis diagnosed within12 hrs of onset for laparoscopic surgery. High insufflation pressure should be avoided as this could push intra abdominal bacteria into blood stream causing bactremia and septic shock. Pneumoperitonium pressure should be below 11 mm of Hg.

TECHNIQUE OF LOCATING THE PERFORATION

After creation of pneumoperitoneum and insertion of trocars (usually four ports) using triangulation concept, laparoscopy is performed. The gall bladder, which usually adheres to the perforation, is retracted by surgeon’s left hand and moved upward.

The gall bladder is passed to the assistant using the sub xiphoid port, which is placed, to the right of falciform ligament area. The perforation is clearly identified as a hole on the anterior aspect of duodenum which has been covered by the fundus of the gall bladder.

CLEANING THE ABDOMEN:

Thorough irrigation and suction of all intra abdominal fluid is done with several litres of saline with local antibiotics. Trial of electrolysed strong acid acquous solution, which contains active oxygen and active chlorine and possesses a redox potential was used for lavage in a study.

Each quadrant is cleaned methodically with special attention to vesicorectal pouch. Fibrous membranes are removed as much as possible.

CLOSURE OF THE PERFORATION:

An intra corporeal technique by inserting omental patch in the knot is preferable than to use the tails of knot to fix the patch (follows the same rule as with original open Graham’s patch)

## OMENTOPLASTY WITH BIOLOGICAL GLUE

Instead of suturing, biological glues (eg. fibrogel) can be applied along the edges of perforation and over this a segment of omentum is held in place for several minutes by atraumatic grasping forceps.

During Laparoscopic perforation closure, using intra corporeal suturing in a manner identical to open surgery, depending on the experience of surgeon, proximal gastric vagotomy or Taylor's procedure may be performed.

## INDICATIONS FOR DEFINITIVE ULCER SURGERY

- Hemodynamically stable young patients
- Perforations < less than 24 hours
- No obvious co-morbidity
- Patients with long history of peptic ulcer
- Perforation of an ulcer during antisecretory agent.
- Previous ulcer complications.
- Anaesthetic surgical facility ideal.
- Combined gastric & duodenal ulcer one of which perforation

- Co existing obstruction and perforation
- Co existing hemorrhage and perforation
- Previous operation for perforated duodenal ulcer

CONTRAINDICTIONS FOR DEFINITIVE ULCER SURGERY

- Associated medical conditions
- Delay in presentation of more than 24 hours
- Gross abdominal contamination with food.

In a followup study, 20.5% patients with acute ulcer perforation treated by simple perforation closures with live omental patch required definitive surgery at a mean of 17.5 months after perforation while 57.5% of for chronic duodenal ulcer perforation required definitive surgery at a mean of 27.4 months after perforation. The introduction of H2 receptor antagonists did not alter the re operation rate.

POST OPERATIVE MANAGEMENT

- Intravenous replacement therapy: The aim of intravenous replacement therapy is to maintain intravascular volume and adequate hydration of the patient that can be monitored by CVP measurement and urinary output.
- Nasogastric drainage: Nasogastric drainage is continued until drainage becomes minimal. At this stage, the nasogastric tube may be removed.
- Antibiotics: the antibiotics commenced preoperatively are continued unless the results of cultures taken at the time of the operation reveal that the causative organisms are resistant to them.
- The goal of antibiotic therapy is to achieve levels of antibiotics at the site of infection that exceed the minimum inhibitory concentrations for the pathogens present.
- In the presence of intra- abdominal infections, gastrointestinal function is often impaired; therefore, oral antibiotics are not efficacious, and intravenous antibiotics are preferred.

- If no obvious improvement in the patient's condition occurs within 2-3 days, the following possibilities are considered.

i. The initial operative procedure was inadequate

ii. Complications have occurred

iii. A super infection has occurred at a new site

iv. The dose of antibiotic is inadequate

v. The antibiotics used do not provide adequate coverage for anaerobes and gram-negative organisms.

- $H_2$ receptor antagonists or proton pump inhibitors for a period of 6 -8 weeks and
- A full regime of H.pylori eradication therapy to be started at the end of 8 weeks.

**COMPLICATIONS**

**EARLY COMPLICATIONS**

- Renal failure and fluid, electrolyte, and pH imbalance
- Respiratory complications.
- Wound infection:

i. Wound infection rates correlate with the bacterial load in the peritoneal fluid.

ii. The judicious use of prophylactic antibiotics has been demonstrated to reduce the incidence of wound infection in contaminated and potentially contaminated wounds.

- Wound failure (partial or total disruption of any or all layers of the operative wound) may occur early (i.e. wound dehiscence)
- The factors are associated with wound failure are malnutrition, sepsis, uremia, diabetes mellitus, corticosteroid therapy, obesity, heavy coughing, hematoma (with or without infection)

- Multiorgan failure and septic shock

i. Septicemia is defined as proliferation of bacteria in the bloodstream resulting in systemic manifestations such as rigors, fever, hypothermia (in gram negative septicemia with endotoxemia), leukocytosis or leucopenia ( in profound septicemia), tachycardia and circulatory collapse.

ii. Septic shock is associated with loss of vasomotor tone, increased capillary permeability, myocardial depression, consumption of WBCs and platelets, dissemination of powerful vasoactive substances, such as histamine, serotonin,

and prostaglandins, resulting in capillary permeability, complement activation and damage of capillary endothelium.

- Gram-negative infections are associated with a much worse prognosis than gram-positive infections, possibly because of associated endotoxemia.
- Localized abdominal abscess
- Entero cutaneous, fistula
- Deep vein thrombosis and pulmonary embolism

LATE COMPLICATIONS

- Mechanical intestinal obstruction: Mechanical obstruction of the intestine is most often caused by postoperative adhesions.
- Incisional hernia
- Recurrent duodenal ulcer
- Haemorrhage (Bleeding duodenal ulcer0

## PATIENTS AND METHODS

This prospective study was conducted is the Department of General Surgery, Government Rajaji Hospital, Madurai Medical College, Madurai for a period of 24 months from July2005 to July 2007.

The patients who presented with duodenal ulcer perforation and underwent surgical management with laparotomy and perforation closure with live omental patch were followed up. The chronicity of the perforated ulcer was decided on basis of history and per operative appearance on peritoneal aspect. All patients were given a course of H.pylori eradication therapy following which H2 receptor blockers or proton pump inhibitors were continued for minimum period of six months.

It was planned to study status of pyloro duodenal canal and ulcers following simple perforation closure by performing UGI endoscopy, about 6 month following perforation closure and / or on patient attending with recurrence of symptoms of duodenal ulcer or its complications.

Endoscopic findings were used as a guide to decide further surgical management as indicated. The definitive surgery done was truncal vagatomy and gastro jejenostomy.

The difficulties encountered during second surgery :

- Dense adhesion between stomach duodenum and liver
- Adherent lesser omentum (fibrosed)
- Duodenum adherent to gall bladder

# DISCUSSION

79 patients who had undergone surgery for perforated duodenal ulcer were selected for this study.

The sex distribution was 73 males and 6 females.

Sex Distribution of Duodenal Perforations

| Sex | No.of patients |
|---|---|
| Male | 73 |
| Female | 6 |

The X ray abdomen showed pneumoperitoneum in 63 patients.

Pneumoperitoneum in Duodenal Perforation

| Gas under diaphragm | No.of cases | Percentage |
|---|---|---|
| Positive | 63 | 79.75 |
| Negative | 16 | 20.25 |

Duodenal perforations were more common in the age group of 30 – 49 years. The youngest case was 18 years and eldest patient was 75 years. The age distribution was as given in table.

Age Distribution of Duodenal Perforation

| S.No. | Age | No.of cases | Percentage |
|---|---|---|---|
| 1. | < 19 | 1 | 1.3 |
| 2. | 22 - 29 | 9 | 11.4 |
| 3. | 30 – 39 | 23 | 29.2 |
| 4. | 40 – 49 | 25 | 31.6 |
| 5. | 50 – 59 | 10 | 12.6 |
| 6. | > 60 | 11 | 13.9 |
| | Total | 79 | 100 |

4 cases had sealed perforation which were reinforced with omental patch. In 5 cases whose general condition did not warrant anaesthesia bilateral flank drainage was done of which 1 patient subsequently undergone perforation closure with omental patch.

For 70 cases simple patch closure of perforation with live omental patch (Graham's patch) was done.

All the patients were advised to continue H2 Blockers or protein pump inhibitor after a course of H pylori eradication therapy and advised to attend review OP regularly for further follow up.

The post operative complication encountered were wound infection (8cases). Right basal pneumonitis 2 cases. Burst abdomen 1 case and enterocutaneous fistula 1 case. The fistula closed spontaneously with conservative management.

Post operative complications in duodenal perforation

| Complications | No.of cases | Percentage |
|---|---|---|
| Wound infection | 8 | 66.66 |
| Basal pneumonitis | 2 | 16.66 |
| Burst abdomen | 1 | 8.33 |
| Entero cutaneous fistula | 1 | 8.33 |

8 patients expired, 5 of whom were the cases treated by flank drainage alone.

Of 15 patients who had 2 years followup, 5 patients had undergone definitive surgery (two in acute duodenal perforation three in chronic duodenal perforation group). Two patients in chronic ulcer group and 1 patient in acute group who was allergic to Ranitidin could not continue protein pump inhibitors, developed gastric outlet obstruction.

Six out of 20 patients in 1 year follow up group required definitive surgery. 1 patient presented with reperforation for whom simple perforation closure followed by definitive ulcer surgery was done. The definitive ulcer surgery done was Truncal vagotomy with gastrojejunostomy except in one patient who had bleeding duodenal ulcer for whom underrunning of ulcer, truncal vagatomy and gastrojejunostomy was done.

## Follow up

| | No.of cases | Acute ulcer group | Chronic ulcer group |
|---|---|---|---|
| 2 years follow up | 15 | 10 | 5 |
| 1 year follow up | 20 | 5 | 15 |
| 6 months follow up | 25 | 20 | 5 |
| No follow up | 11 | - | - |
| Mortality | 8 | - | - |

25 patients reviewed in 6 months follow up and UGI endoscopic study was performed. 18 patients in acute duodenal ulcer perforation group (total 20) and 2 patients in chronic duodenal ulcer group (total 5) had normal study.

2 patients in acute ulcer perforation and 3 patients in chronic duodenal ulcer group had endoscopic features of persisting peptic ulcer disease, for whom definitive ulcer surgery was done. Among the chronic ulcer group one patient had giant duodenal ulcer and another had kissing ulcer.

## Endoscopic follow up

| | Number of patients | Normal | Ulcer + /- complication |
|---|---|---|---|
| 2 years follow up | | | |
| Acute | 10 | 8 | 2 |
| Chronic | 5 | 2 | 3 |
| 1 year follow up | | | |
| Acute | 5 | 4 | 1 |
| Chronic | 15 | 10 | 5 |
| 6 months follow up | | | |
| Acute | 20 | 18 | 2 |
| Chronic | 5 | 2 | 3 |
| No follow up | - | - | - |
| Mortality | - | - | - |

## Surgeries in followup group

| Duration of followup | Ulcer Group | Reason for Surgery | | | |
|---|---|---|---|---|---|
| | | Total No. of patients | Persisting ulcer disease | Gastric outlet obstruction | Others |
| 2 years | Acute | 2 | 1 | 1 | 0 |
| | Chronic | 3 | 1 | 2 | 0 |
| 1 years | Acute | 1 | 1 | 0 | 0 |
| | Chronic | 5 | 1 | 2 | 2 |
| 6 months | Acute | 2 | 2 | 0 | 0 |
| | | 3 | 3 | 0 | 0 |
| Total | | 16 | 9 | 5 | 2 ** |

** one patient had bleeding duodenal ulcer another had reperforation

# CONCLUSION

- Duodenal ulcer perforation was the commonest cause of gastrointestinal perforation with a male preponderance
- More common in the fourth decade of life
- More common in the lower socio-economic class of people
- Smoking and alcohol were aggravating factors
- Perforation was the first manifestation of peptic ulcer disease in a small percentage of patients.
- The role of nonsteroidal anti-inflammatory drugs as the cause of acute perforation was noticed in this study group.
- Radiological evidence of pneumoperitoneum could not be established in nearly one third of the patients.
- Inspite of recent advances is closing duodenal perforation by laparoscopy, simple closure with omental patch was widely practiced in the study group.
- Simple closure with omental patch with thorough peritoneal toileting was very much effective

- Definitive ulcer surgery was not warranted in the emergency and treatment with $H_2$ blockers and H. pylori eradication achieved good control over the disease in the follow up period, especially in acute perforation group.
- Segregation of group into acute or chronic is done on the basis of history, per operative appearance of ulcer site on peritoneal aspect which may need further refinement. (Application of diagnostic tests for H.pylori)
- Endoscopic follow up can help to identify sub group of patients needing definitive surgery earlier
- Most of the patients with chronic duodenal ulcer perforation require definitive surgery for ulcer.
- Follow up should be for a period of 2 years in case of acute duodenal ulcer perforation and for 3 years is case of chronic duodenal ulcer perforation.

# BIBLIOGRAPHY


1. Shakelford's, Surgery of Alimetary tract, George D. Zudema M.D., Charles J. Yeo. MD., Fifth edition, vol II, chapter 2, 16-22.
2. Farquharson's text books of operative general sugery, Margaret Farquharson, FRCS Ed. Bredan Moran FRCSI nineth edition, chapter 16, 258-260.
3. Sir Alfred Cuschieri Essentials Surgical Practice – Disorders of stomach and duodenum, 4th edition, module 8, 271.
4. Forbes GM, Glaster ME, Cullen DJ et al, Duodenal ulcer treated with H.pylori eradication – seven years follow up, Lancet 1994 ; 343, 258-60.
5. Laine L. The long term management of patients with bleeding ulcers. H.Pylori eradication instead of maintenance antisecretory therapy. Gastro intest Endosco : 41 : 77-79.
6. Sung JY, Chung SCS, Leung VKS et al, H. Pylori eradication in maintenance H2 Blockade in preventing recurrent ulcer bleeding, Gastro intest Endosco 1996 : 43: 359.

7. Feldman M. Peptic ulcer perforation into the lesser peritoneal sac. I.A Statistical study of 57 collected cases. Am J. Dig. Dis Sci. 1950 ; 17 : 333-334.
8. Sturup J Raahave D . Retroperitoneal perforation in duodenal ulcer. Ann Chir Gynaecol. 1985 ; 74 : 299-300.
9. Norris J R ., Haubrisch WS : The incidence and clinical features of penetration in peptide ulceration JAMA 1961 ; 178: 386.
10. Mark Feld mann, Lawrence, S Friedman et al. Fordlerans Gastrointestinal and liver disease 7th edition, Vol I, 770.
11. Paster SR, Brogdon BG : Roentgenographic diagnosis of pneumoperitoneum ; JAMA 1976 ; 235 : 1264-1268.
12. Svanes C ; Salvesen H, Bjerke – Larssen T et al, Trends and value and consequences of radiologic imaging of perforated gastro duodenal ulcer – A, 50 year experience. Scand J Gastro enterology 1990, 25 : 257 – 262.
13. Apple Gate GR, Braunstein P, Wang FL Radionuclede detection of duodenal ulcer perforation, J Nucl Med 1989 May 30(5) 700-702.

14. Shaheen A, Becker SG, Sandler M, Perforated anterior duodenal ulcer, diagnsosis with Technium, $^{99m}$ Tc DISIDA South Med J 1987 80(3) 394-396.
15. Crofts TJ, Park KG, Stede RJ et al – A randomized trials of non operative treatment for perforated peptic ulcer, N Engl J Med 1989 ; 320 : 970-973.
16. Keane TE, Dillon B, Afdhal et al, Conservative management of perforated duodenal ulcer, Br J Surg 1988 ; 75 ; 583-584.
17. Stringer MD, Cameron AE Surgeons's attitudes to the operative management of duodenal ulcer perforation and haemorrhage, Ann R coll Surg Eng 1988, July 70(4), 220-223.
18. Lace WY, Leung KL, Chung SCS et al, Laparoscopic repair of Perforated Peptic Ulcer, Br J Sur 1995 ; 82 : 814-816.
19. Inone Y, Endo S, Kondo K et al, Trial of Electrolysed strong solutions in treatment of peritonitis and intraperitoneal abscess, Artif organs 1997 Jan : 21(1) : 28-31.

20. De Watteville JC, Testas PL. Laparoscopy in intestinal emergencies, Laparascopic diagetive surgery (Ed) L. Testas Bernaud J Delaite, 1994 ; 16 : 146-147.
21. Gallen P, Ryon W Peel AL et al, Duodenal ulcer perforation – the effect of H2 antagonist. Ann R Coll Surg. Engl. 1986, Sep ; 68 (5) : 240-242.

# PROFORMA

Name : IP No. :

Age/ Sex : Occupation :

Address :

Date of admission : Date of Surgery :

Date of Discharge :

PRESENTING COMPLAINTS :

22. Pain Abdomen :
23. Abdominal distension:
24. Constipation :
25. Obstipation :
26. Vomiting :
27. Others :

HISTORY OF PRESENT ILLNESS :

I - PAIN ABDOMEN

a) Mode of onset :
b) Duration :
c) Site :
d) Radiation :
e) Nature :
f) Aggravating :
Relieving factor :

II - ABDOMINAL DISTENTION

a) Duration :
b) Frequency :

c) Character :

d) Vomitus :

e) Relation to pain :

IV - CONSTIPATION / OBSTIPATION

V - OLIGURIA

VI – OTHERS

PAST HISTORY

1. Ulcer pain :
2. Haemetemesis :
3. Melaena :
4. Previous surgery :

PERSONAL HISTORY

1. Smoker :
2. Alcoholic :
3. Diet :

MENSTURAL HISTORY

Last menstrual period :

EXAMINATION

1. GENERAL EXAMINATION

| | | | |
|---|---|---|---|
| Consciousness | : | Attitude | : |
| Hydration | : | Appearance: | |
| Pulse | : | Blood pressure | : |
| Respiratory rate | : | Temperature | |
| Anemia | : | Jaundice | |

II – EXAMINATION OF THE ABDOMEN

INSPECTION

a) Shape :

b) Skin :

c) Scar :

d) Swelling :

e) Umblicus :

f) Respiratory movements:

g) Peristaltic movements :

h) Hernial sites :

PALPITATION

a) Temperature :

b) Tenderness :

c) Guarding :

d) Rigidity :

e) Mass :

f) Liver / spleen :

PERCUSSION

a) Liver dullness :

b) Shifting dullness :

AUSCULTATION

Bowel sounds

EXTERNAL GENITALIA

DIGITAL RECTAL EXAMINATION

a) Cardiovascular system

b) Respiratory system

c) Central nervous system

INVESTIGATIONS

1. Blood grouping
2. Haemoglobin

3. Blood sugar
4. Blood urea
5. Serum creatinine
6. Plain x ray abdomen erect
7. Ultrasound abdomen

PREVIOUS SURGERY DETAILS :

CLINICAL DIAGNOSIS :

MANAGEMENT :

Conservative / Operative

Preoperative resuscitation

IV fluid / Blood transfusion

OPERATIVE NOTES :

Date :

Anesthesia :

Peroperative Findings :

FINAL DIAGNOSIS

POST OPERATIVE COMPLICATIONS

FOLLOW UP ENDOSCOPIC DIAGNOSIS

MANAGEMENT

Date :

Anesthesia :

Pre operative preparations :

Surgery :

# DUODENUM FIRST PART - LARGE ACUTE ON CHRONIC DUODENAL ULCER

**PYLORIC ORIFICE TOTALLY OCLUDED WITH HEALED ULCERS AROUND – STRICTURE PYLORUS**

**ESOPHAGUS – Multiple Longitudinal inflamed areas present**

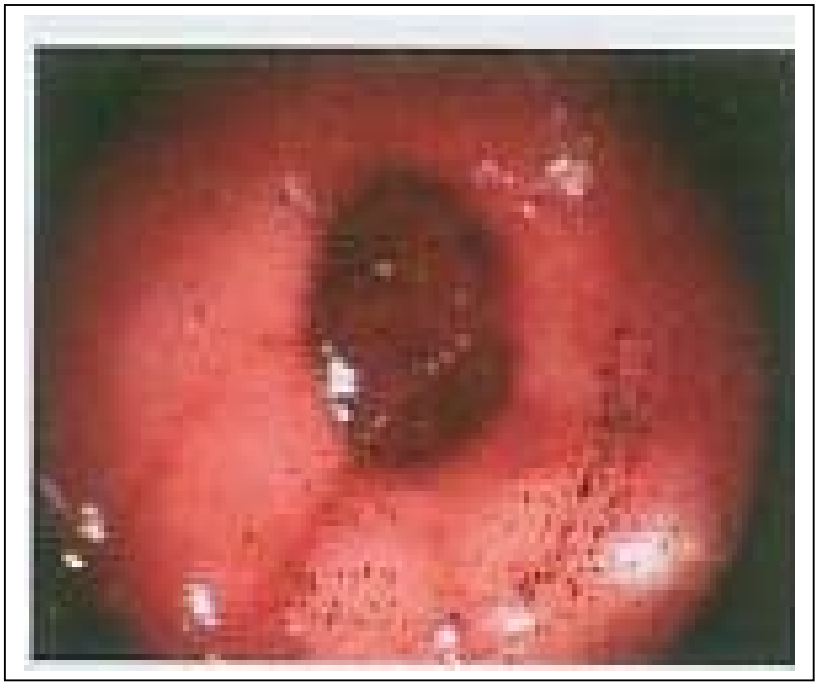
**Stomach – Scattered areas of inflammation present throughout**

**Pyloroduodenal orifice narrowed – Duodenum not entered**

**Esophagitis, Pangastritis, Chronic duodenal ulcer with gastric outlet obstruction**

# GASTRIC OUTLET OBSTRUCTION

BLEEDING DUODENAL ULCER

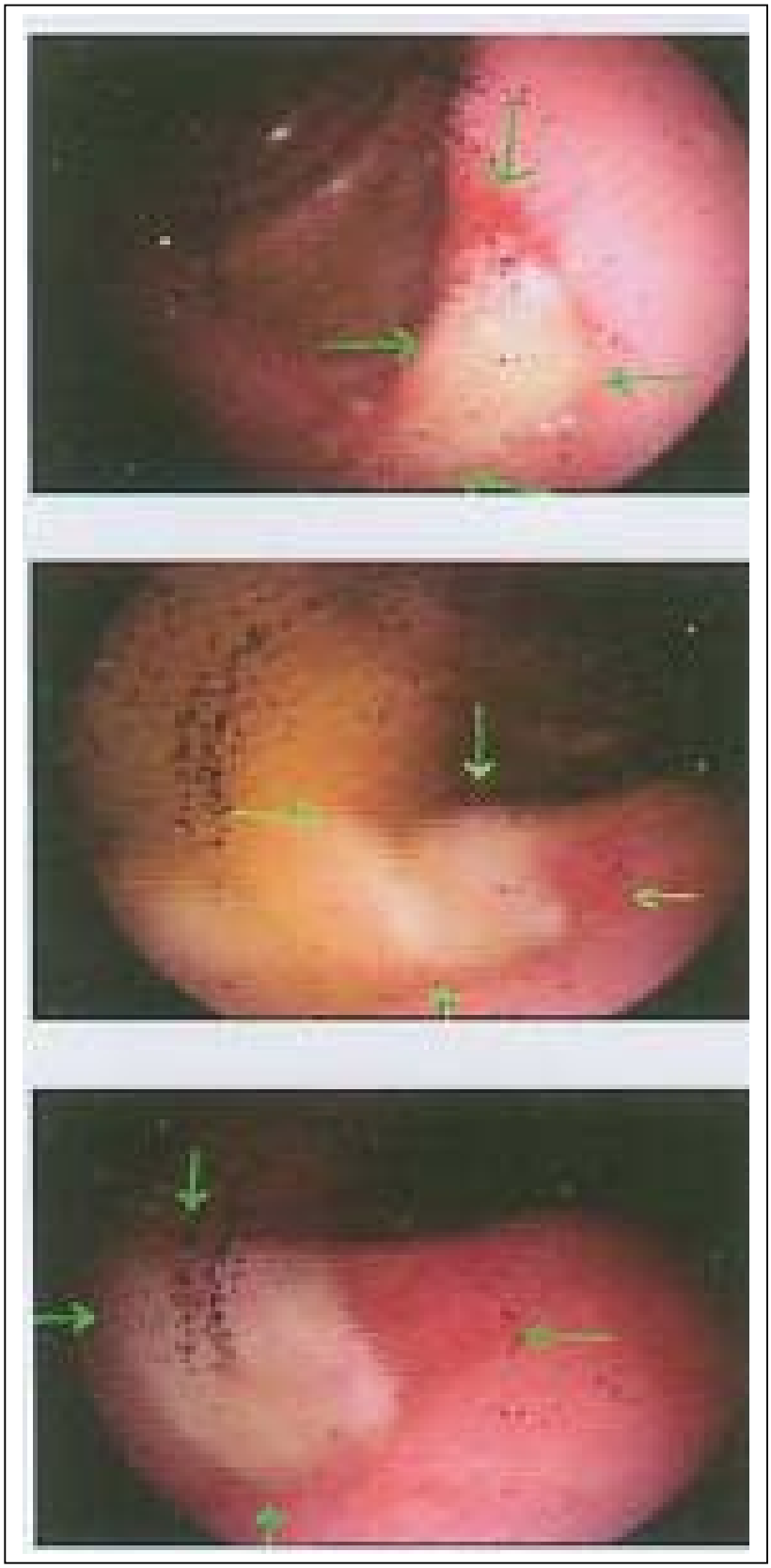


STOMAL ULCER WITH EDEMA

EFFERENT LOOP STOMA EDEMATOUS

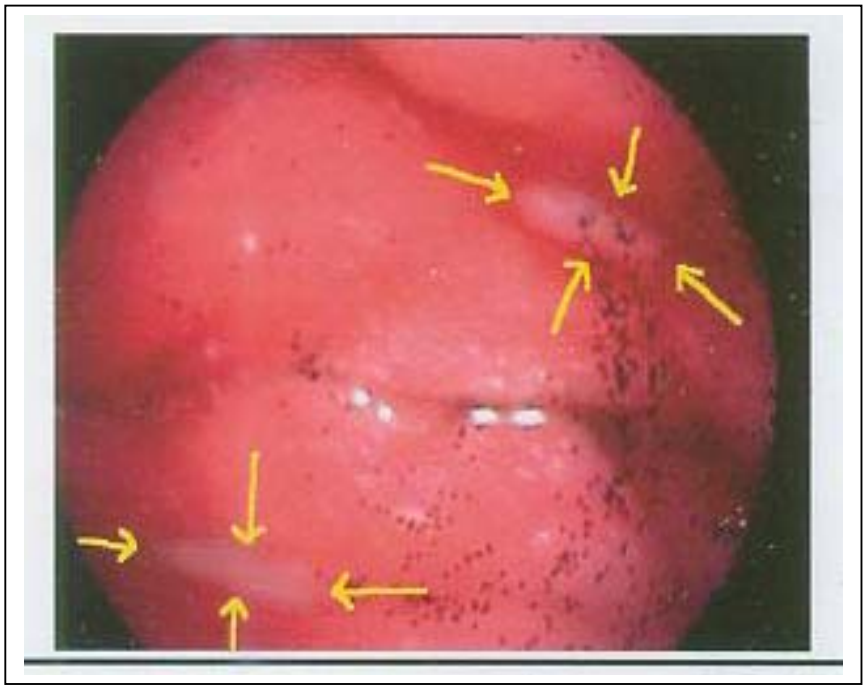
AFFERENT LOOP NORMAL

ANASTOMOTIC ULCER

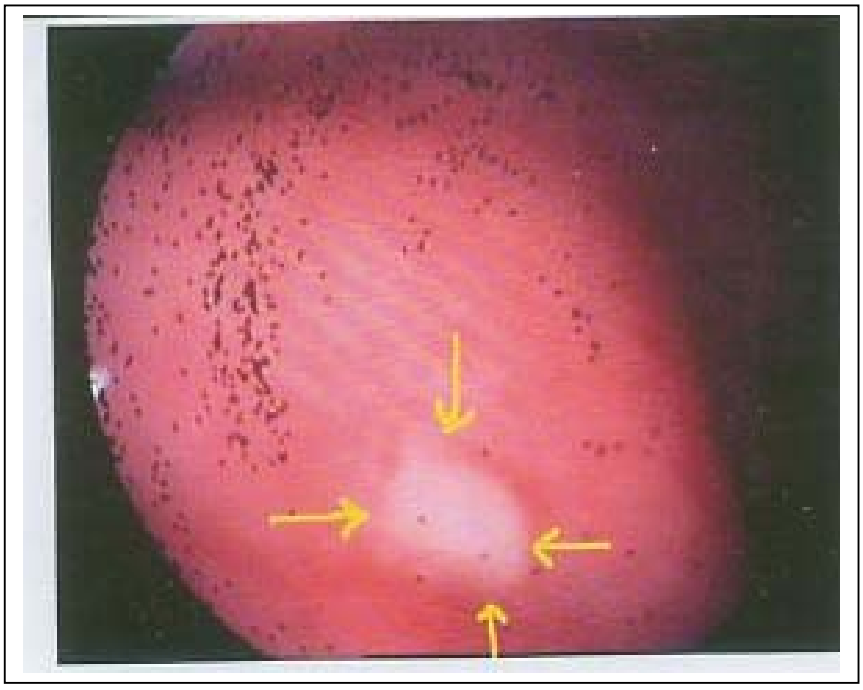


## DUODENAL ULCER

## KISSING ULCER - DUODENUM

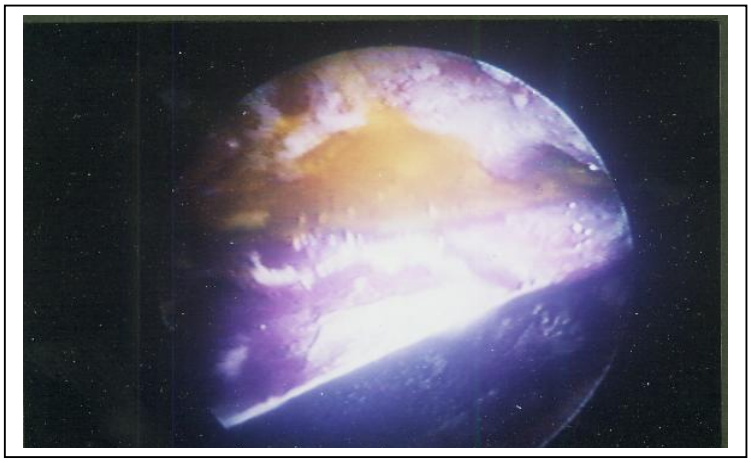


DUODENAL ULCER

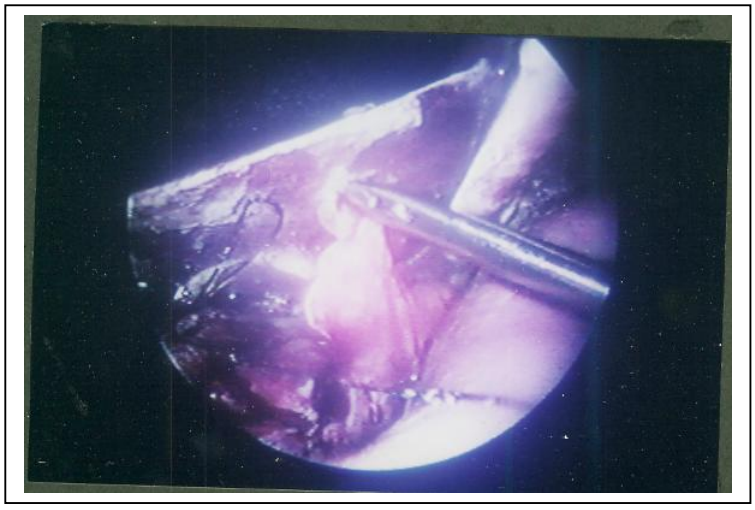


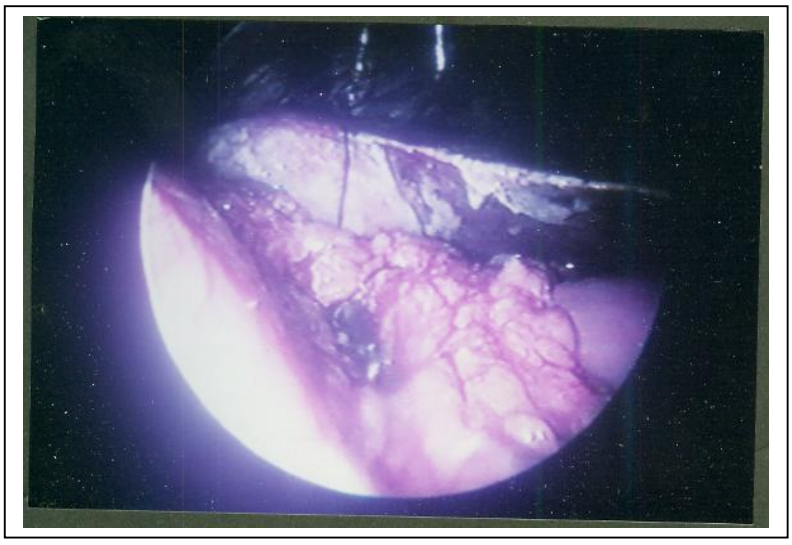
**GIANT DUODENAL ULCER**

LAPROSCOPIC REPAIR OF DUODENAL ULCER PERFORATION
BILE LEAK SEEN

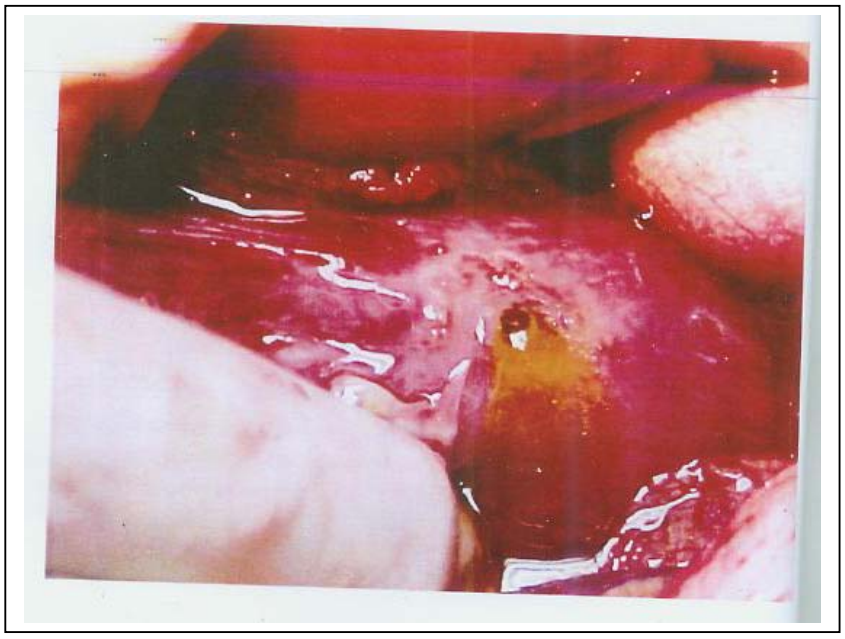


OMENTAL PATCH REPAIR





DUOPENAL ULCER PERFORATION



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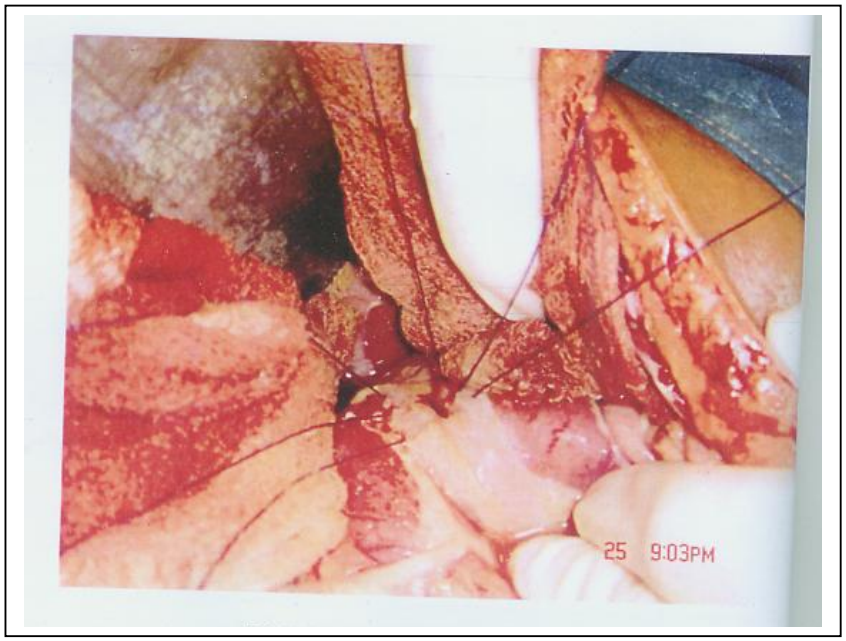

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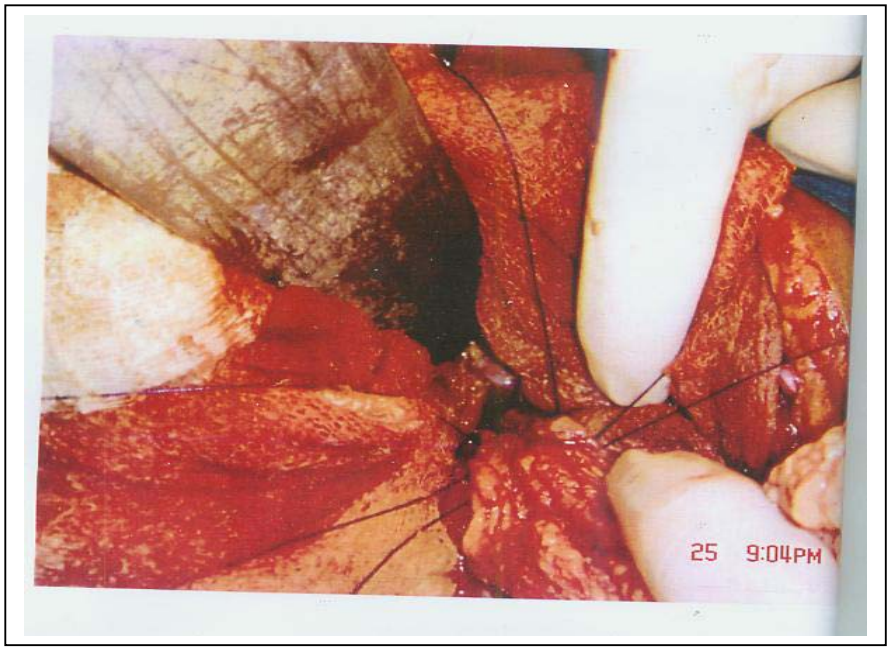
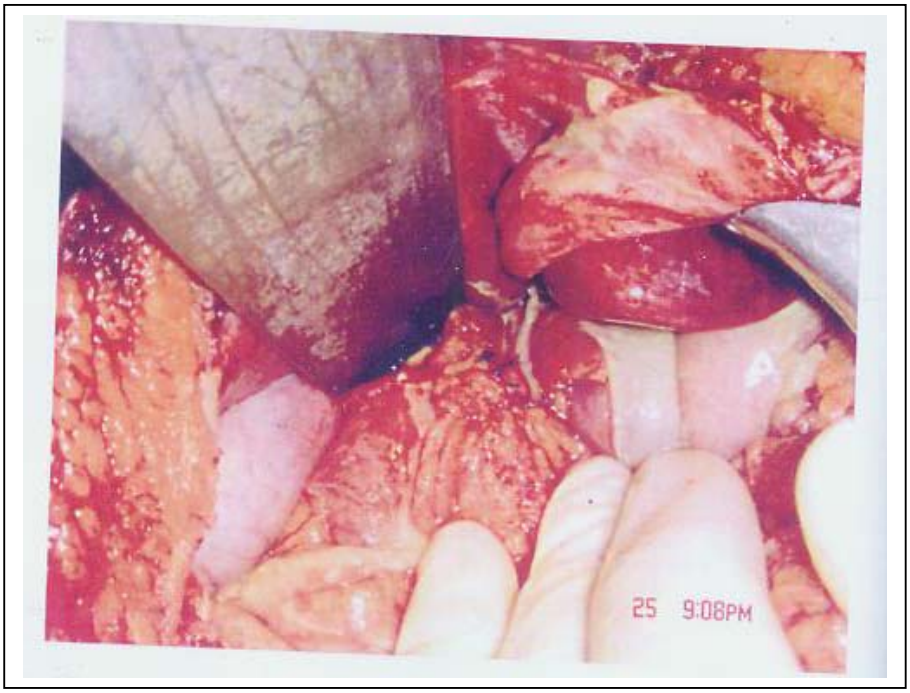
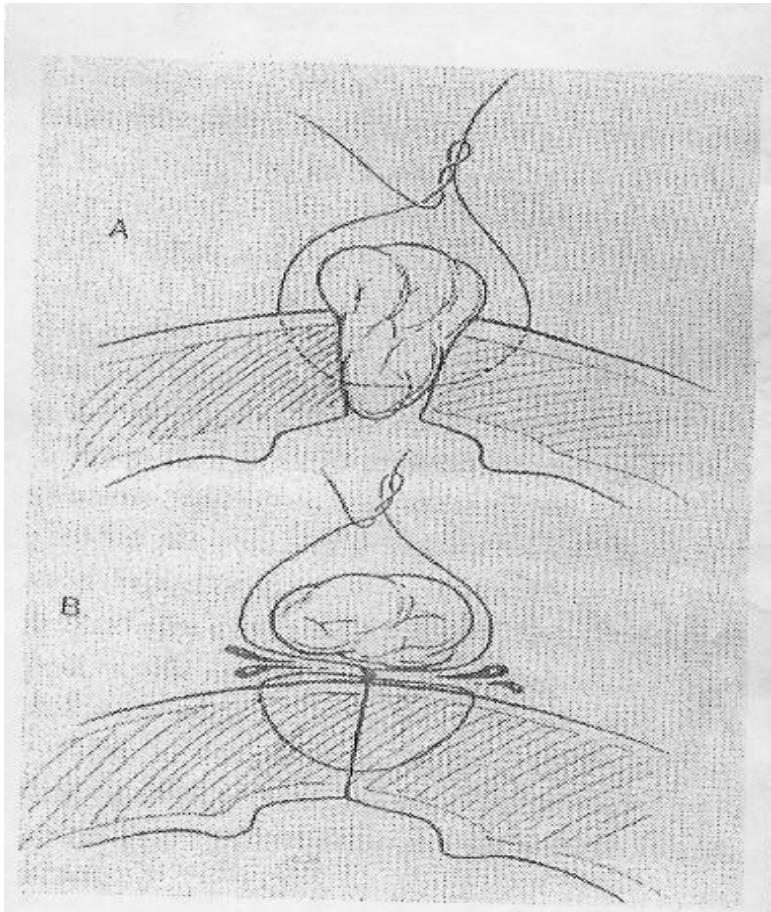



OMENTAL PATCH REPAIR COMPLETED



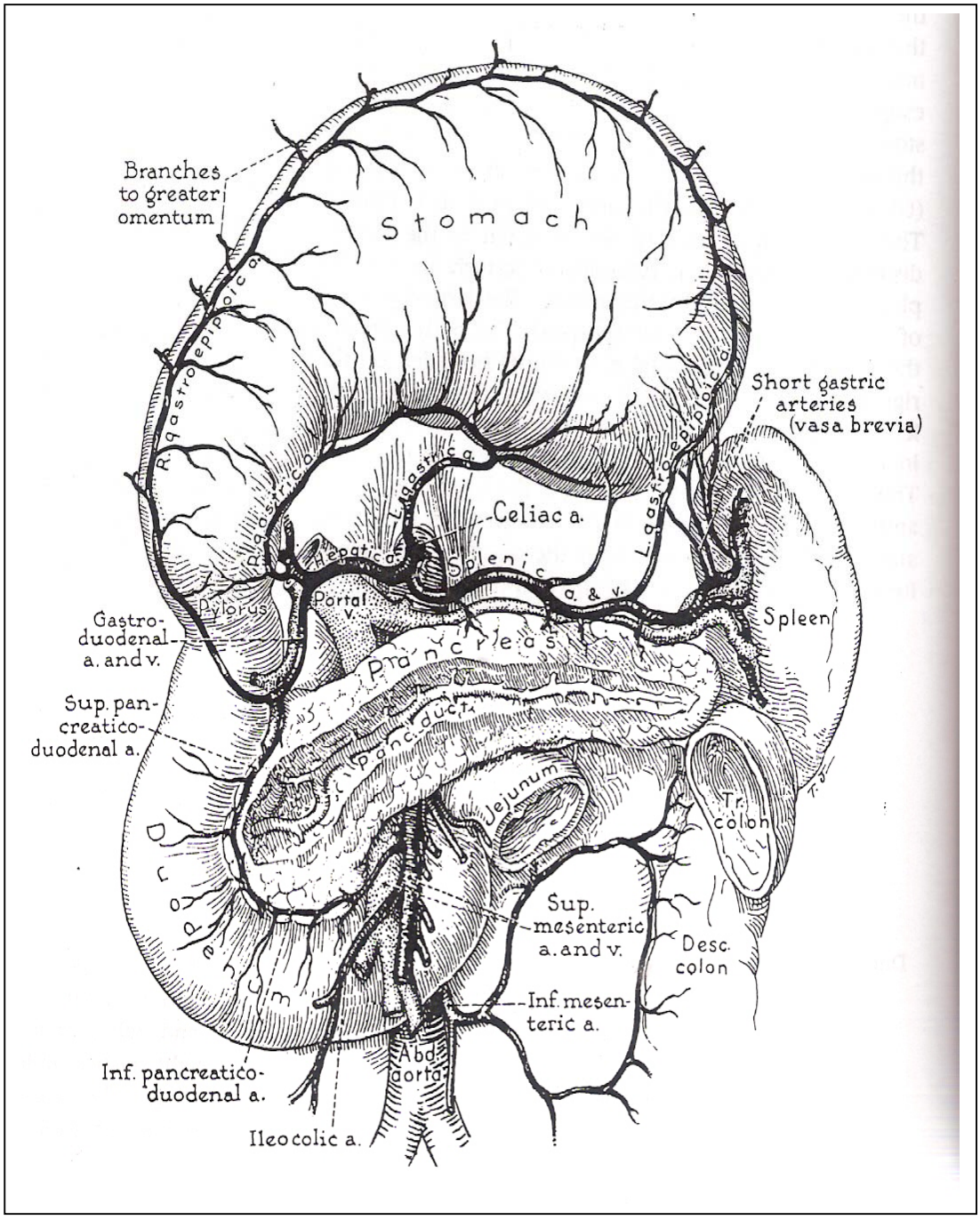
# OMENTAL PATCH CLOSURE



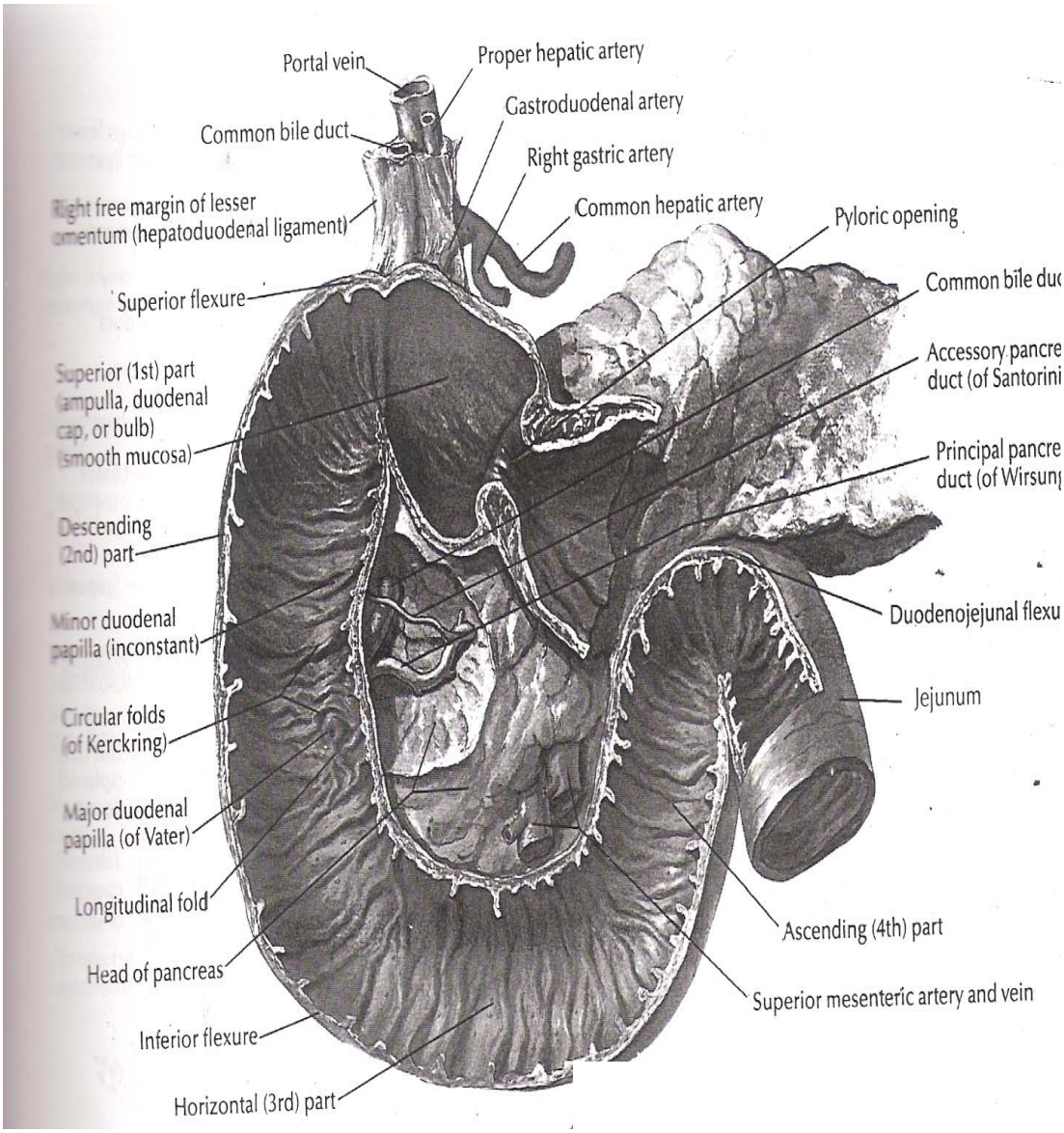

B = Less efficacious

# ARTERIAL BLOOD SUPPLY TO THE STOMACH AND DUODENUM

(Stomach reflected upward and pancreatic duct is exposed)



# DUODENUM - ANATOMY



# MASTER CHART

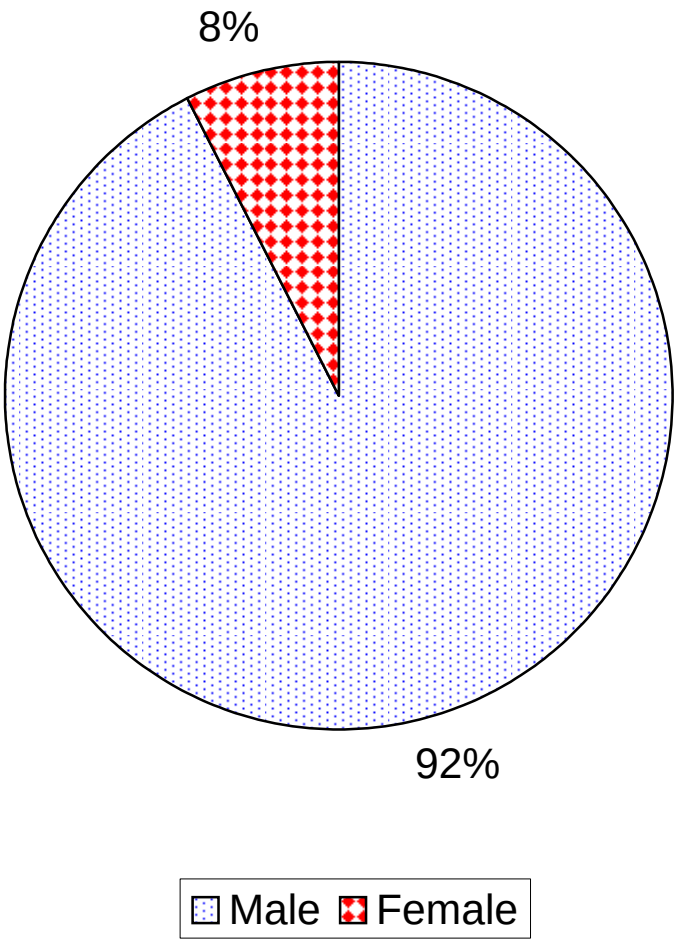
| S.No. | Name | IPNo. | Age | Sex | Chronicity of Ulcer | Surgery done | Follow up endoscopy | Definitive surgery |
|---|---|---|---|---|---|---|---|---|
| 1. | Vellachami | 3881 | 45 | M | Acute | Omental patch closure | - | - |
| 2. | Palanisamy | 4596 | 33 | M | Acute | Omental patch closure | Active D.U. | Truncal vago tomy and GJ |
| 3. | Arul | 6424 | 57 | M | Chronic | Omental patch closure | Active D.U. | Truncal vago tomy and GJ |
| 4. | Ramachandran | 4958 | 45 | M | Acute | Omental patch closure | Normal | - |
| 5. | Shanmugam | 8077 | 45 | M | Chronic | Omental patch closure | Normal | - |
| 6. | Poothaiammal | 8889 | 30 | F | Acute | Omental patch closure | Gastric outlet obstruction | Truncal vago tomy and GJ |
| 7. | Nazir | 10080 | 60 | M | - | Flank drain done | - | - |
| 8. | Muniyapandi | 10585 | 32 | M | Acute | Omental patch closure | Normal | - |
| 9. | Subramani | 13729 | 40 | M | Acute | Omental patch closure | Normal | - |
| 10. | Vishvendra | 14105 | 42 | M | Acute | Omental patch closure | - | - |
| 11. | Ramanathan | 17354 | 75 | M | Chronic | Omental patch closure | - | - |
| 12. | Ramasamy | 17601 | 48 | M | Chronic | Reinforcement with omental patch | - | - |
| 13. | Mahalingam | 18926 | 55 | M | Chronic | Flank drain F/B perforation closure | Normal | - |
| 14. | Pandi | 17098 | 45 | M | Chronic | Omental patch closure | Gastric outlet obstruction | Truncal vago tomy and GJ |
| 15. | Pandian | 20618 | 60 | M | Chronic | Omental patch closure | Normal | - |
| 16. | Ravichandran | 23709 | 35 | M | Acute | Omental patch closure | Normal | - |
| 17. | Chinnaponnu | 28277 | 32 | F | Acute | Omental patch closure | Normal | - |
| 18. | Marutha muthu | 27207 | 32 | M | Chronic | Omental patch closure | Normal | - |
| 19. | Murugesan | 30372 | 32 | M | Acute | Omental patch closure | Normal | - |
| 20. | Ramesh | 31328 | 25 | M | Acute | Omental patch closure | Normal | - |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 21. | Chinnasamy | 34465 | 26 | M | Chronic | Reinforcement with omental patch | Normal | - |
| 22. | Palaniammal | 39957 | 42 | F | Acute | Omental patch closure | Normal | - |
| 23. | Karuppasamy | 40166 | 25 | M | Acute | Omental patch closure | Normal | - |
| 24. | Vadivel | 43790 | 28 | M | Acute | Omental patch closure | Normal | - |
| 25. | Rani | 44096 | 37 | F | Acute | Omental patch closure | Normal | - |
| 26. | Vellaiyan | 44958 | 62 | M | Chronic | Omental patch closure | - | - |
| 27. | Narayanan | 45558 | 45 | M | Acute | Omental patch closure | Normal | - |
| 28. | Muthusamy | 47262 | 75 | M | Chronic | Omental patch closure | Normal | - |
| 29. | Periya pagavan | 469930 | 70 | M | Chronic | Omental patch closure | - | |
| 30. | Selvam | 471315 | 38 | M | Acute | Omental patch closure | Normal | - |
| 31. | Ganesan | 472282 | 43 | M | Chronic | Omental patch closure | Normal | - |
| 32. | Veeranam | 405645 | 45 | M | Acute | Omental patch closure | D.U. | Truncal vago tomy and GJ |
| 33. | Vellaiyammal | 475636 | 60 | F | Chronic | Omental patch closure | - | |
| 34. | Periyasamy | 476388 | 50 | M | Chronic | Omental patch closure | G.O.O. | Truncal vago tomy and GJ |
| 35. | Mookkan | 477258 | 45 | M | Acute | Omental patch closure | Normal | - |
| 36. | Andisamy | 477322 | 42 | M | Acute | Reinforcement with omental patch | Normal | - |
| 37. | Maniyados | 477899 | 70 | M | Acute | Omental patch closure | - | - |
| 38. | Murugan | 478128 | 20 | M | Acute | Omental patch closure | Normal | - |
| 39. | Chinadurai | 479248 | 35 | M | Acute | Omental patch closure | Normal | - |
| 40. | Kalimuthu | 479488 | 30 | M | Acute | Omental patch closure | Normal | - |
| 41. | Veeran | 479468 | 60 | M | Chronic | Omental patch closure | - | |
| 42. | Kumar | 479468 | 36 | M | Chronic | Omental patch closure | Normal | - |
| 43. | Pitchaiammal | 479871 | 40 | F | Chronic | Omental patch closure | G.O.O. | Truncal vago tomy and GJ |
| 44. | Veeranan | 484477 | 40 | M | Acute | Omental patch closure | Normal | - |
| 45. | Karuppiah | 484882 | 30 | M | Acute | Omental patch closure | Normal | - |
| 46. | Radhakrishnan | 484955 | 39 | M | Chronic | Omental patch closure | Normal | - |
| 47. | Senthilkumar | 485337 | 30 | M | Acute | Omental patch closure | Normal | - |

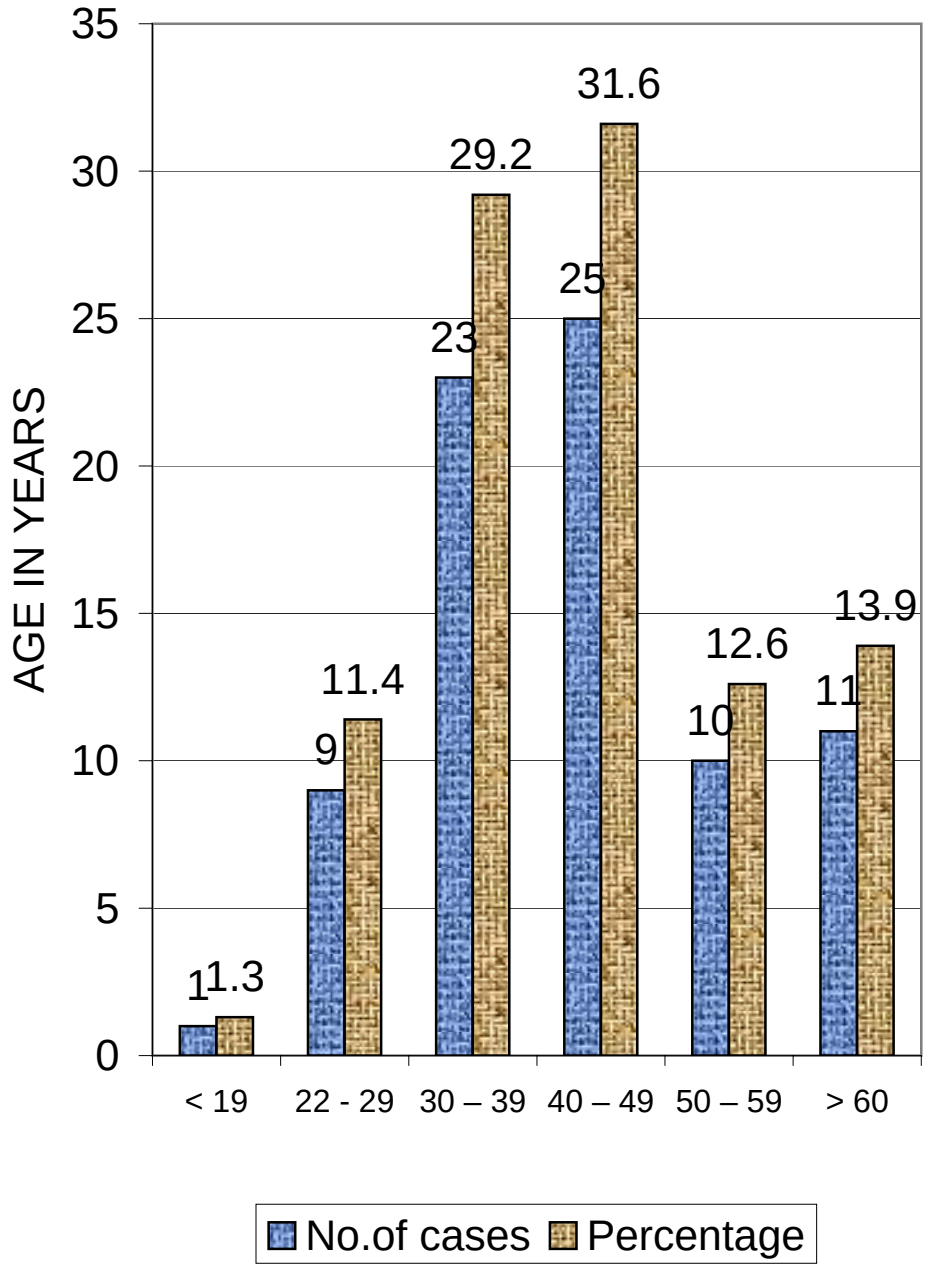
| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 48. | Selvi | 486160 | 30 | F | Chronic | Omental patch closure | - | - |
| 49. | Pandi | 487067 | 45 | M | Acute | Omental patch closure | - | |
| 50. | Irulandi | 487205 | 50 | M | Chronic | Omental patch closure | Normal | - |
| 51. | Muthulingam | 487444 | 36 | M | Acute | Omental patch closure | Normal | - |
| 52. | Karuppasamy | 487665 | 36 | M | Chronic | Reinforcement with omental patch | Normal | - |
| 53. | Baskar | 488920 | 46 | M | Chronic | Omental patch closure | Bleeding D.U. | Under running of ulcer & TV C.J. |
| 54. | Chellaiah | 489080 | 63 | M | Acute | Omental patch closure | - | - |
| 55. | Maduraiveeran | 489380 | 28 | M | Acute | Omental patch closure | Normal | - |
| 56. | Pathammal | 497022 | 35 | F | Acute | Omental patch closure | Normal | - |
| 57. | Manikkam | 492795 | 22 | M | Acute | Omental patch closure | - | - |
| 58. | Mariappan | 493955 | 50 | M | Chronic | Omental patch closure | Active D.U. | Truncal vago tomy and GJ |
| 59. | Vairam | 495313 | 35 | M | Chronic | Omental patch closure | | |
| 60. | Chinnakaruppan | 495599 | 50 | M | Acute | Omental patch closure | Active D.U. | Truncal vago tomy and GJ |
| 61. | Kandaraj | 496555 | 45 | M | Chronic | Omental patch closure | Normal | - |
| 62. | Kulanthaivelu | 496872 | 55 | M | Chronic | Omental patch closure | Normal | - |
| 63. | Raj | 497222 | 49 | M | Chronic | Omental patch closure | - | - |
| 64. | Yacoop | 497459 | 42 | M | Chronic | Omental patch closure | Active D.U. | Truncal vago tomy and GJ |
| 65. | Sethuraman | 497782 | 50 | M | Acute | Omental patch closure | Normal | - |
| 66. | Suresh | 498190 | 34 | M | Chronic | Omental patch closure | - | - |
| 67. | Muthu | 498458 | 40 | M | Chronic | Omental patch closure | Normal | - |
| 68. | Jeyaraj | 498731 | 45 | M | - | Flank drain | - | - |
| 69. | Malairaj | 498789 | 40 | M | Chronic | Reperforation closure | Active D.U | Truncal vago tomy and GJ |
| 70. | Raman | 501084 | 55 | M | Acute | Omental patch closure | Normal | - |
| 71. | Karuppan | 501144 | 63 | M | - | Flank drain | - | - |
| 72. | Mayee | 501167 | 55 | M | Chronic | Omental patch closure | Normal | - |
| 73. | Ganesan | 503261 | 38 | M | Chronic | Omental patch closure | Normal | - |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 74. | Karthick | 563533 | 18 | M | Chronic | Omental patch closure | Normal | - |
| 75. | Andisamy | 1003/07 | 30 | M | Chronic | Omental patch closure | Active D.U. | Truncal vago tomy and GJ |
| 76. | Malai karuppan | 2290 | 27 | M | Acute | Omental patch closure | Active D.U. | Truncal vago tomy and GJ |
| 77. | Ramamoorthy | 2863 | 40 | M | Chronic | Omental patch closure | Active D.U. | Truncal vago tomy and GJ |
| 78. | Kannadaiyan | 3230 | 35 | M | - | Flank drain | - | - |
| 79. | Shanmugavel | 5520 | 45 | M | Chronic | Omental patch closure | Gastric outlet obstruction | Truncal vago tomy and GJ |

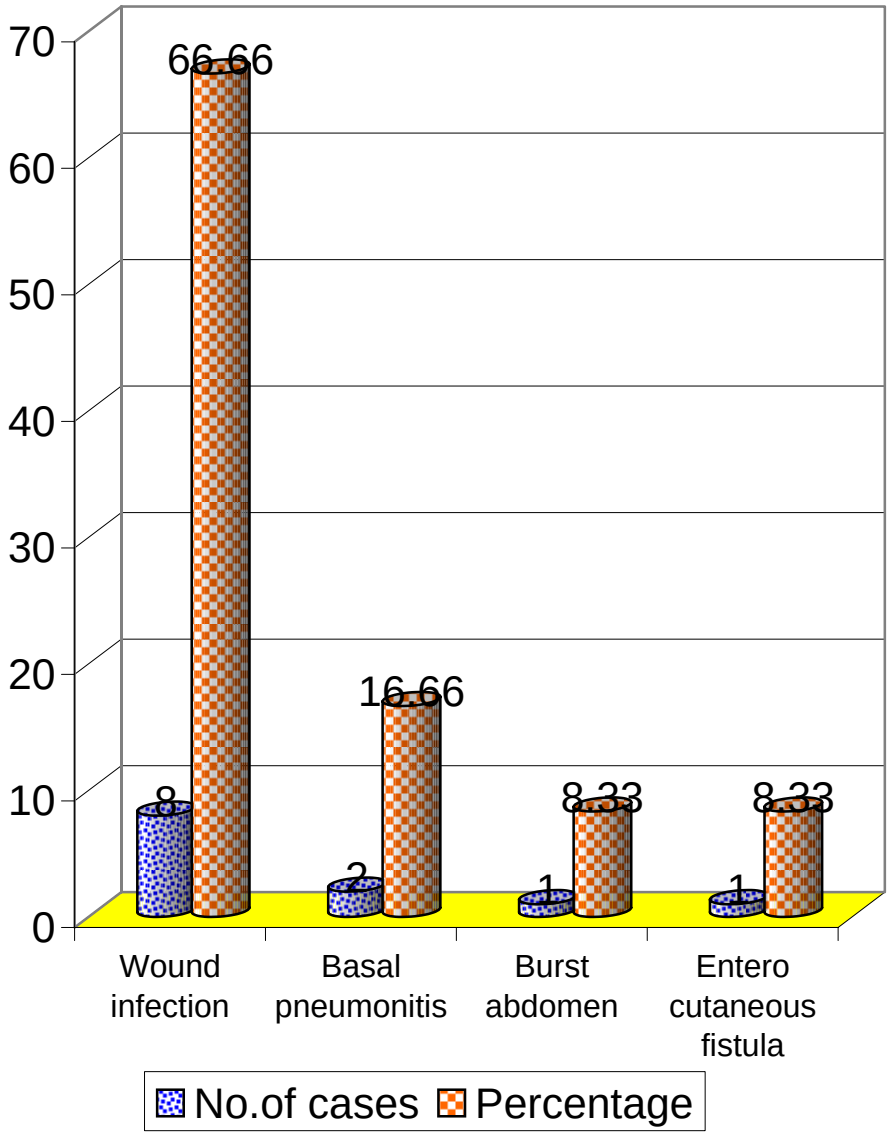
## SEX DISTRIBUTION



## AGE DISTRIBUTION



## POST OPERATIVE COMPLICATIONS



| | |
|---|---|
| Male | 73 |
| Female | 6 |

| Age | No.of cases | Percentage |
|---|---|---|
| < 19 | 1 | 1.3 |
| 22 - 29 | 9 | 11.4 |
| 30 – 39 | 23 | 29.2 |
| 40 – 49 | 25 | 31.6 |
| 50 – 59 | 10 | 12.6 |
| > 60 | 11 | 13.9 |

| Complications | No.of cases | Percentage |
|---|---|---|
| Wound infection | 8 | 66.66 |
| Basal pneumonitis | 2 | 16.66 |
| Burst abdomen | 1 | 8.33 |
| Entero cutaneous fistula | 1 | 8.33 |

| Gas under diaphragm | No.of cases | Percentage |
|---|---|---|
| Positive | 63 | 79.75 |
| Negative | 16 | 20.25 |